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Chronic Calcific Pancreatitis Presenting With Secondary Diabetes In A Young Adult

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ABSTRACT:

Chronic calcific pancreatitis (CCP) is a progressive inflammatory disorder of the pancreas characterized by fibrosis, atrophy, and calcifications, leading to both exocrine and endocrine dysfunction. Patients commonly present with chronic abdominal pain, nausea, vomiting, weight loss, steatorrhea, and secondary diabetes (type 3c diabetes mellitus) due to pancreatic endocrine insufficiency. Diagnosis relies on clinical evaluation, laboratory tests, and imaging, including ultrasound, CECT, MRI/MRCP, and endoscopic ultrasound. Management is multidisciplinary, involving pain control, pancreatic enzyme replacement, insulin therapy, nutritional support, and, in selected cases, endoscopic or surgical interventions such as lateral pancreatic jejunostomy. Appropriate treatment improves pain, pancreatic drainage, glycaemic control, and nutritional status, although CCP remains a chronic condition requiring long-term follow-up.

KEYWORDS: Chronic Calcific Pancreatitis, Type 3c Diabetes Mellitus, Endocrine Dysfunction, Lateral Pancreatic jejunostomy

INTRODUCTION:

Chronic calcific pancreatitis (CCP) is a progressive inflammatory disorder of the pancreas that results in fibrosis and calcifications within the gland. [1]This chronic damage can affect both the exocrine and endocrine functions of the pancreas, including destruction of insulin-producing beta cells. One of the significant complications of CCP [2]is secondary diabetes, known as type 3c diabetes mellitus (T3cDM), which arises due to impaired pancreatic endocrine function. This case report highlights a presentation of CCP with secondary diabetes, emphasizing its clinical manifestations, diagnostic considerations, and implications for management. As a result, damage to the pancreas reduces insulin production, leading to impaired blood glucose control. This condition is called secondary diabetes, as it develops due to an underlying pancreatic disease. [3,4]Chronic calcific pancreatitis usually presents with chronic upper abdominal pain, which may radiate to the back, along with nausea, vomiting, weight loss, and fatty stools due to impaired pancreatic digestion. Over time, damage to the pancreas can lead to secondary diabetes, causing high blood sugar, excessive thirst, frequent urination, and fatigue.

Risk factors include chronic alcohol use, smoking, genetic predisposition, recurrent pancreatitis, and pancreatic duct obstruction. The condition can result in complications such as exocrine insufficiency, [5,6]persistent pain, malnutrition, pancreatic calcifications, pseudocysts, biliary obstruction, and an increased risk of pancreatic cancer, all of which may significantly affect the patient's quality of life.

Type 3c diabetes mellitus (T3cDM), or pancreatic diabetes, often develops in patients with chronic pancreatitis (CP), [7]especially in those with alcohol-related disease and pancreatic calcifications. Diabetes occurs due to damage to insulin-producing cells, leading to high blood sugar and symptoms like excessive thirst, hunger, and urination.

Secondary diabetes can also result from other conditions, including hormonal disorders, cystic fibrosis, or certain medications. In CP, the risk of diabetes increases with the severity and duration of pancreatic inflammation.[8,9]

Hormonal disorders can also contribute to secondary diabetes by disrupting insulin production or action. Conditions such as acromegaly, hyperthyroidism, and pheochromocytoma can increase the risk of developing diabetes.[9,10]

Certain medications may trigger secondary diabetes by affecting insulin secretion or increasing insulin resistance. [11,12]Glucocorticoids, used to treat inflammatory conditions, can raise blood sugar levels, and some antipsychotics and immunosuppressants have been associated with an increased risk.[13,14]

The development of secondary diabetes is influenced by multiple factors, and not every individual with these conditions will develop diabetes. However, these factors significantly increase the risk, emphasizing the need for careful monitoring and appropriate management in affected individuals.

CASE PRESENTATION:

An 18-year-old male, a known case of Type 2 Diabetes Mellitus for the past two months, presented with complaints of right lower abdominal pain for the last three months. The pain was sudden in onset, gradually progressive, intermittent, non-radiating, and aggravated on lying in the supine position. It was associated with vomiting containing food particles. The patient had a previous history of similar complaints and was admitted earlier for uncontrolled blood glucose levels.

On admission, the patient was diagnosed with chronic calcific pancreatitis with uncontrolled high blood sugar levels. His vital parameters were within normal limits; however, blood glucose levels were markedly elevated (Fasting Blood Sugar – 146 mg/dl, Postprandial – 465 mg/dl, HbA1c – 11.4%).

On examination, percussion revealed liver dullness, and bowel sounds were present on auscultation.

CECT Abdomen showed a diffusely atrophic pancreas with multiple parenchymal calcifications, the largest measuring 9×9 mm in the head region. The main pancreatic duct was dilated, measuring approximately 6–7 mm in calibre, with intraductal calcifications. The gall bladder appeared partially distended.

The findings were suggestive of chronic calcific pancreatitis. The patient was planned for lateral pancreatic jejunostomy as part of the surgical management plan.

Under general anaesthesia, the patient was positioned supine, and a midline vertical incision was made to enter the abdominal cavity. The lesser sac was exposed by dividing the gastrocolic ligament, revealing an atrophic pancreas. The main pancreatic duct was identified and opened, followed by a 5 cm longitudinal incision along its course. A lateral pancreatic jejunostomy was performed by anastomosing the proximal jejunal loop end-to-side and the distal loop side-to-side, ensuring adequate ductal drainage. Haemostasis was secured, a drain was placed, and the wound was closed in anatomical layers.

Initially, the patient's elevated blood glucose levels were managed with Insulin therapy: Inj. Human Act rapid (11U-11U-8U) and Inj. Lantus (0-0-30U) for 2 days. Following the surgical procedure, the patient received postoperative medications, including Inj. Diclofenac aqueous 1 amp in 100 ml NS for pain, Inj. Sulbactam + Etoperidone 1.5 gm and Inj. Metronidazole 500 mg as an antibiotic, Inj. Octreotide 100 mcg to reduce pancreatic secretions, and Inj. Tramadol 1 amp in 100 ml NS for additional analgesia.

Postoperatively, serosanguineous drainage was observed from the surgical drain, indicating a mixture of blood and serous fluid, which is a common and expected finding during the early postoperative period, reflecting normal wound healing.

DISCUSSION:

Chronic calcific pancreatitis (CCP) is a progressive pancreatic disorder that can lead to both exocrine and endocrine dysfunction. This case highlights the development of type 3c diabetes mellitus in a young patient with CCP, emphasizing how pancreatic damage and calcifications impair insulin production. Patients typically present with chronic abdominal pain, vomiting, weight loss, and steatorrhea, as seen in this patient. Risk factors such as alcohol use, genetic predisposition, and recurrent pancreatitis increase the likelihood of disease progression and secondary diabetes. Management of CCP requires a multidisciplinary approach, including pain control, insulin therapy, pancreatic enzyme replacement, and surgical interventions like lateral pancreatic jejunostomy in cases with ductal dilatation. Postoperative care, including monitoring for drainage and glycaemic control, is essential to ensure recovery and prevent complications.

CONCLUSION:

This case demonstrates that chronic calcific pancreatitis can present with secondary diabetes even in young patients. It highlights the importance of early recognition, careful monitoring, and a combined medical and surgical approach. Lateral pancreatic jejunostomy effectively relieves ductal obstruction and helps improve pancreatic drainage, pain, and overall patient outcomes, while insulin therapy manages the associated hyperglycaemia. Long-term follow-up is essential to monitor pancreatic function and prevent further complications.

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